Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application:

In the claims:

1. (original) A method of diagnosing Alzheimer's Disease in a mammal, the method comprising contacting an ocular tissue with a detectably-labeled compound that binds to an amyloid protein, wherein the compound is a fluorescent dye, wherein an increase in binding of said compound to said ocular tissue compared to a normal control level of binding indicates that said mammal is suffering from or is at risk of developing Alzheimer's Disease.

2. (original) The method of claim 1, wherein said compound is thioflavin S.

3. (original) The method of claim 1, wherein said compound is thioflavin T.

4. (original) The method of claim 1, wherein said detectably-labeled compound preferentially binds to an amyloid–β (Aβ) polypeptide.

5. (original) The method of claim 4, wherein said detectably-labeled compound preferentially binds to Aβ (1-42).

6. (original) The method of claim 1, wherein the method further comprises imaging a cortical region of the eye.

7. (original) The method of claim 6, wherein the method further comprises imaging a supranuclear region of an eye.

8. (original) The method of claim 1, wherein said increase is at least 10% greater than said normal control value.

9. (original) The method of claim 1, wherein said increase is at least 25% greater than said normal control value.

10. (original) The method of claim 1, wherein said increase is at least 50% greater than said normal control value.

11. (original) The method of claim 1, wherein said increase is at least 100% greater than said normal control value.

12. (original) A method of diagnosing Alzheimer's Disease in a mammal, the method comprising contacting an ocular tissue with a detectably-labeled compound that binds to an amyloid protein, wherein the compound is detectably-labeled with a positron emitting radionuclide and wherein an increase in binding of said compound to said ocular tissue compared to a normal control level of binding indicates that said mammal is suffering from or is at risk of developing Alzheimer's Disease.

13. (original) The method of claim 12, wherein said positron-emitting radionuclide is selected from the group consisting of Carbon 11, Nitrogen 13, Oxygen 15, and Fluorine 18.

14. (original) The method of claim 12, wherein said detectably-labeled compound is a Chrysamine compound.

15. (original) The method of claim 12, wherein said detectably-labeled compound preferentially binds to an amyloid–β (Aβ) polypeptide.

16. (original) The method of claim 15, wherein said detectably-labeled compound preferentially binds to Aβ (1-42).

17. (original) The method of claim 12, wherein the method further comprises imaging a cortical region of the eye.

18. (original) The method of claim 17, wherein the method further comprises imaging a supranuclear region of an eye.

19. (original) The method of claim 12, wherein said increase is at least 10% greater than said normal control value.

20. (original) The method of claim 12, wherein said increase is at least 25% greater than said normal control value.

21. (original) The method of claim 12, wherein said increase is at least 50% greater than said normal control value.

22. (original) The method of claim 12, wherein said increase is at least 100% greater than said normal control value.

23. (original) A method of diagnosing Alzheimer's Disease in a mammal, the method comprising contacting an ocular tissue with a detectably-labeled compound that binds to an amyloid protein, wherein the compound is detectably-labeled with a radioactive label and wherein an increase in binding of said compound to said ocular tissue compared to a normal control level of binding indicates that said mammal is suffering from or is at risk of developing Alzheimer's Disease.

24. (original) The method of claim 23, wherein said radioactive label is selected from the group consisting of ^{3}H and ^{125}I.

25. (original) The method of claim 23, wherein said detectably-labeled compound is a Chrysamine compound.

26. (original) The method of claim 23, wherein said detectably-labeled compound preferentially binds to an amyloid–β (Aβ) polypeptide.

27. (original) The method of claim 26, wherein said detectably-labeled compound preferentially binds to Aβ (1-42).

28. (original) The method of claim 24, wherein the method further comprises imaging a cortical region of the eye.

29. (original) The method of claim 28, wherein the method further comprises imaging a supranuclear region of an eye.

30. (original) The method of claim 24, wherein said increase is at least 10% greater than said normal control value.

31. (original) The method of claim 24, wherein said increase is at least 25% greater than said normal control value.

32. (original) The method of claim 24, wherein said increase is at least 50% greater than said normal control value.

33. (original) The method of claim 24, wherein said increase is at least 100% greater than said normal control value.

34. (original) A method of diagnosing Alzheimer's Disease in a mammal, the method comprising using magnetic resonance imaging to determine an amount of an amyloid protein present in an ocular tissue, wherein an increase in the amount of said amyloid protein of said amyloid protein present in the ocular tissue compared to the amount of amyloid protein present in a normal ocular tissue indicates that said mammal is suffering from or is at risk of developing Alzheimer's Disease.

35. (original) The method of claim 34, wherein said amyloid protein is an amyloid–β (Aβ) polypeptide.

36. (original) The method of claim 35, wherein amyloid-β (Aβ) polypeptide is Aβ (1-40).

37. (original) The method of claim 35, wherein amyloid-β (Aβ) polypeptide is Aβ (2-40).

38. (original) The method of claim 35, wherein amyloid-β (Aβ) polypeptide is Aβ (1-42).

39. (original) The method of claim 34, wherein the magnetic resonance imaging is performed on a cortical region of the eye.

40. (original) The method of claim 39, wherein the magnetic resonance imaging is performed on a supranuclear region of an eye.

41. (original) A method of diagnosing Alzheimer's Disease in a mammal, the method comprising using magnetic resonance imaging to determine an anatomical location of an amyloid protein present in an ocular tissue, wherein a difference in the anatomical location of said amyloid protein present in the ocular tissue compared to the anatomical location present in a normal ocular tissue indicates that said mammal is suffering from or is at risk of developing Alzheimer's Disease.

42. (original) The method of claim 41, wherein said amyloid protein is an amyloid–β (Aβ) polypeptide.

43. (original) The method of claim 42, wherein amyloid-β (Aβ) polypeptide is Aβ (1-40).

44. (original) The method of claim 42, wherein amyloid-β (Aβ) polypeptide is Aβ (2-40).

45. (original) The method of claim 42, wherein amyloid-β (Aβ) polypeptide is Aβ (1-42).

46. (original) The method of claim 41, wherein the magnetic resonance imaging is performed on a cortical region of the eye.

47. (original) The method of claim 46, wherein the magnetic resonance imaging is performed on a supranuclear region of an eye.

48. (original) The method of claim 41, wherein the anatomical area in the ocular tissue of the subject is the supranuclear region of an eye

49. (original) A method for prognosis of Alzheimer's Disease, the method comprising:

a) contacting ocular tissue of a mammal with a compound which binds to an amyloid polypeptide.

b) quantitating the level of association of said compound with said ocular tissue; and

c) comparing the level of association with a normal control level of association, wherein increasing levels of association over time indicates an adverse prognosis,

wherein the quantitating is accomplished by positron emission tomography, radioimaging, radioimmunoassay, or magnetic resonance imaging.

50. (New) A method of diagnosing a prionopathy or a predisposition thereto in a mammal, comprising

(a) contacting an ocular tissue with a detectably-labeled compound, which preferentially binds to an amyloid protein located in said ocular tissue;

(b) allowing said compound to distribute into the lens; and

(c) imaging said ocular tissue,

wherein said detectably-labeled compound comprises a fluorophor, wherein said detectably-labeled compound is lipophilic, and wherein an increase in binding of said compound to said ocular tissue compared to a normal control level of binding indicates that said mammal is suffering from or is at risk of developing said prionopathy.

51. (New) The method of claim 50, wherein said prionopathy is selected from the group consisting of scrapie, bovine spongiform encephalopathy, Creutzfeld-Jakob disease, variant Creutzfeld-Jakob disease, and spongiform encephalopathies.

52. (New) A method for prognosis of a prionopathy, comprising

(a) contacting ocular tissue of a mammal with a compound which preferentially binds to an amyloid polypeptide, wherein said compound comprises a fluorophor and wherein said detectably-labeled compound is lipophilic;

(b) allowing said compound to distribute into the lens

(c) imaging said ocular tissue;

(d) quantitating the level of association of said compound with said ocular tissue; and

(e) comparing said level of association with a normal control level of association, wherein increasing levels of association over time indicates an adverse prognosis.

53. (New) The method of claim 52, wherein said prionopathy is selected from the group consisting of scrapie, bovine spongiform encephalopathy, Creutzfeld-Jakob disease, variant Creutzfeld-Jakob disease, and spongiform encephalopathies.